Weed Technology. 2005. Volume 19:683–696

Potato Variety Tolerance to Flumioxazin and Sulfentrazone[1]

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Abstract: Field studies were conducted at Aberdeen, ID; Ontario, OR; and Paterson, WA, to evaluate potato tolerance to flumioxazin and sulfentrazone. In 'Russet Burbank' tolerance trials conducted in 2000 at ID, OR, and WA, sulfentrazone applied preemergence (PRE) at rates ranging from 105 to 280 g ai/ha caused significant injury consisting of stunting, leaf discoloration-blackening, and/or leaf malformation-crinkling at 4 wk after treatment (WAT). By 12 WAT, injury was $\leq$5%. At 4 WAT, flumioxazin applied PRE at 105 and 140 g ai/ha resulted in injury, whereas 53 g ai/ha did not cause significant injury. At 12 WAT, no visual injury was present at the ID site, whereas flumioxazin at 140 g/ha was still causing injury in WA. Regardless of initial injury, Russet Burbank tuber yields at ID, OR, and WA were not reduced as a result of any flumioxazin or sulfentrazone treatment compared with the nontreated controls. In potato variety tolerance trials conducted at ID in 2000 and at WA in 2002 with Russet Burbank, 'Ranger Russet', 'Russet Norkotah', and 'Shepody' and at ID in 2002 with those varieties plus 'Alturas' and 'Bannock Russet', early season injury caused by flumioxazin or sulfentrazone applied PRE at rates as high as 210 g ai/ha or 280 g/ha, respectively, occurred, but variety tuber yields were not reduced compared with nontreated control yields. In contrast, at ID in 2001, early injury caused by flumioxazin or sulfentrazone applied PRE at 105 or 210 g/ha translated to tuber yield reductions of all six varieties tested compared with the nontreated controls. At WA in 2001, Ranger Russet tuber yields were reduced by PRE applications of flumioxazin at 53 to 140 g/ha or sulfentrazone at 105 to 280 g/ha, and Shepody total tuber yields were reduced by all rates of PRE-applied sulfentrazone. Russet Burbank and Russet Norkotah tuber yields were unaffected by either herbicide. Unusual heat stress occurring early in the 2001 growing season at both locations may have compounded the effects of herbicide injury and, consequently, tuber yields were reduced in 2001, whereas injury occurring in 2000 or 2002 during relatively normal growing conditions did not translate to yield reductions.

Nomenclature: Sulfentrazone; flumioxazin, potato, *Solanum tuberosum* L. 'Alturas', 'Bannock Russet', 'Ranger Russet', 'Russet Burbank', 'Russet Norkotah', 'Shepody'.

Additional index words: Crop safety, herbicide injury, potato variety tolerance.

Abbreviations: ID, Idaho; O.M., organic matter; OR, Oregon; PNW, Pacific Northwest; PRE, preemergence; protox, protoporphyrinogen oxidase; WA, Washington; WAT, weeks after treatment.

INTRODUCTION

Flumioxazin and sulfentrazone are newly registered herbicides for use in soybean [*Glycine max* (L.) Merr.] (Anonymous 2003, 2004b) and are being developed for use in potato to control hairy nightshade (*Solanum sarrachoides* Sendter) and other broadleaf weeds (Boydston et al. 2001; Kazarian et al. 2001; Tonks et al. 2001; Wilson et al. 2002). Sulfentrazone was recently registered for use in potato (Anonymous 2004b, 2004c). Applied preemergence (PRE), these herbicides also can control acetolactate synthase (ALS) and triazine herbicide-resistant weed biotypes (Boydston et al. 2001; Taylor-Lovell et al. 2002). Flumioxazin is an N-phenylphthalmide derivative, and sulfentrazone is an aryl triazinone (Vencill 2002a, 2002b), and both inhibit protoporphyrinogen oxidase (protox) (protoporphyrin IX:oxygen oxidoreductase, EC 1.3.3.4), an enzyme important in the chlorophyll biosynthetic pathway, resulting in light-induced membrane lipid peroxidation (Dayan and Duke 1997; Duke et al. 1991; Vencill 2002a, 2002b). This mode of

[1] Received for publication July 22, 2004, and in revised form October 22, 2004. University of Idaho Agricultural Experiment Station Paper 04P02.

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action is different from that of other potato herbicides (Wilson et al. 2002). After application to susceptible plants, toxic intermediates, such as porphyrins, accumulate, photosensitization occurs, and membrane disruption and lipid peroxidation is initiated.

Flumioxazin and sulfentrazone can be taken up in roots or foliage (Vencill 2002a, 2002b). After soil application, most susceptible plants die as they begin to emerge, and the remaining die shortly after exposure to sunlight. Foliar contact to susceptible plants results in rapid desiccation and necrosis of exposed plant tissues. Sulfentrazone has been shown to induce electrolyte leakage from excised roots of germinating soybean seedlings (Li et al. 2000a). Other manifestations of sulfentrazone injury also have occurred in the absence of light, such as reduced root hair elongation in coffee senna (*Cassia occidentalis* L.) (Dayan et al. 1996) and reduced hypocotyl length in soybean (Li et al. 1999). Because of these observations, Li et al. (2000a) have speculated that sulfentrazone may have an additional or secondary mode of action separate from protox inhibition.

Variability in sulfentrazone tolerance has been attributed to differential metabolism (Dayan et al. 1996), differential root absorption (Wehtje et al. 1997), differential absorption in early stages of growth (Li et al. 2000b), localized differences in soil pH influencing root absorption (Ferrell et al. 2003), sulfentrazone availability as influenced by soil cation-exchange capacity (Kerr et al. 2004), and differential tolerance to peroxidative stress caused by the herbicide (Dayan et al. 1997). Other researchers have proposed that because protox is located in shoot tissue, and translocation to shoots is essential for sulfentrazone toxicity, differential root absorption and differential translocation of sulfentrazone from roots to shoots are the primary mechanisms of differential tolerance (Bailey et al. 2003).

Similar to sulfentrazone, some research results indicate that differences in flumioxazin tolerance are due to differential metabolism (Price et al. 2004c). However, Taylor-Lovell et al. (2001) have stated that because soybean varieties most sensitive to flumioxazin were different from those sensitive to sulfentrazone, tolerance mechanisms may be different for the two herbicides even though they have the same mode of action. Price et al. (2004a) suggest that differential cotton (*Gossypium hirsutum* L.) tolerance to flumioxazin is due to differential absorption, translocation, or metabolism at various growth stages and the development of a bark layer.

Soil loss of both herbicides is primarily by microbial degradation and the half-life for flumioxazin is shorter than for sulfentrazone (11.9 to 17.5 d compared with 121 to 302 d, respectively) (Vencill 2002a, 2002b). Because sulfentrazone is a weak acid with pKa of 6.56, at pH levels greater than the pKa, sulfentrazone would exist in a greater ratio of anionic to molecular (uncharged) forms. Regardless of pH, flumioxazin does not dissociate. In studies using selected soils with pH ranging from 5.1 to 7.8 and organic matter (O.M.) from 1.1 to 4.4%, flumioxazin sorption was 100% in all soils tested whereas sulfentrazone had greater sorption for soils with a pH similar or less than its pKa than for soils with a higher pH. (Kazarian et al. 2001).

Grey et al. (1997) also observed sulfentrazone sorption was most affected by soil pH because sulfentrazone sorption generally decreased as soil pH increased, with the greatest decrease in sorption occurring in pH above the pKa of sulfentrazone. Sulfentrazone availability and crop injury increases with high pH, low O.M., and coarse soil texture, especially under high moisture conditions (Grey et al. 1997; Krausz and Young 2003; Niekamp et al. 1999; Swantek et al. 1998; Taylor-Lovell et al. 2001; Wehtje et al. 1997). The potential for soybean phytotoxicity from flumioxazin also increases with high soil moisture and low organic matter content (Sakaki et al. 1991; Taylor-Lovell et al. 2001).

White, red, yellow, and purple potato cultivars were not differentially sensitive to sulfentrazone rates as high as 425 g ai/ha in a Washington trial conducted on a silt loam soil with pH 5.9 and 3.3% O.M. (Miller and Libbey 2002). Results from Colorado field trials conducted in a sandy loam soil with pH 7.3 and 1.1% O.M. indicated good tolerance of Russet Norkotah, 'Russet Nugget', 'Chipeta', and 'Sangre' potato varieties to flumioxazin applied PRE at rates ranging from 35 to 70 g ai/ha. Chipeta, however, was injured by sulfentrazone applied PRE at 280 g/ha, and Sangre was injured by PRE sulfentrazone applications ranging from 140 to 280 g/ha (Kazarian et al. 2001; Wilson et al 2002). Although in-season injury occurred, tuber yields of herbicide-treated potato plants were not reduced in those trials compared with nontreated controls regardless of herbicide, rate, or variety.

'Superior' potato was tolerant to sulfentrazone applied PRE at rates as high as 280 g/ha in Virginia trials conducted on a sandy loam soil with pH 6.2 and <1% O.M. (Bailey et al. 2002). 'Atlantic' was tolerant to sulfentrazone applied PRE at rates ranging from 110 to 280 g/ha in Texas trials conducted on a loamy sand soil with pH 7.6 and 0.6% O.M. and on a fine sand soil with pH 7.0 and 0.6% O.M. but not in a trial conducted on a fine

Table 1. Planting, preemergence herbicide application, and harvest dates for potato tolerance trials conducted at Aberdeen, ID, Ontario, OR, and Paterson, WA, in 2000, 2001, and 2002 with flumioxazin and sulfentrazone.

				Harvest	
Location	Year	Planting date	Application date	Date	Days after planting
Aberdeen, ID					
Russet Burbank trial	2000	May 02	May 26	Sept. 21	142
Variety trial	2000	May 02	May 26	Sept. 21	142
Variety trial	2001	May 08	June 01	Sept. 26	141
Variety trial	2002	May 08	June 03	Oct. 01	146
Ontario, OR					
Russet Burbank trial	2000	April 19	May 09	Oct. 16	180
Paterson, WA					
Russet Burbank trial	2000	April 18	May 10	Sept. 27	172
Variety trial	2001	April 03	May 02	Aug. 20	139
Variety trial	2002	March 27	April 24	Sept. 12	170

sand soil with pH 6.8 and 0.5% O.M. (Grichar et al. 2003). The researchers stated that the significant potato crop injury and yield reductions caused by sulfentrazone applied PRE at 110 to 280 g/ha at the latter site may be the result of irrigation occurring 48 h after application and also because of the coarse soil (91% sand) at that site, whereas the two other sites had 81 or 85% sand, respectively.

Whereas russet potato varieties have been tolerant to sulfentrazone and flumioxazin in Colorado (sandy loam, pH 7.3, 1.1% O.M.), Russet Burbank has exhibited unacceptable crop injury from sulfentrazone rates greater than 210 g/ha in previous ID studies conducted on a loam soil with pH 7.9 and 1.3% O.M. (Tonks et al. 2001). Differential potato variety tolerance to herbicides is not unusual because some varieties, such as chipping (e.g., Chipeta), red (e.g., Sangre), and white-skinned cultivars (e.g., Atlantic, Shepody, Superior), express sensitivity to metribuzin whereas russet varieties, such as Russet Burbank, Russet Norkotah, and Russet Nugget, are more tolerant (Callihan and Eberlein 1991).

Russet Burbank, Russet Norkotah, Ranger Russet, and Shepody are the four major potato varieties planted in the United States, accounting for approximately 46, 12, 9, and 7%, respectively, of U.S. hectarage in 2002 (Anonymous 2004a). The objective of these studies was to determine crop response of these four potato varieties, and two new varieties being grown in the Pacific Northwest (PNW), Bannock Russet and Alturas, to PRE applications of flumioxazin and sulfentrazone.

MATERIALS AND METHODS

Russet Burbank tolerance trials were conducted in 2000 at the University of Idaho Aberdeen Research and Extension Center near Aberdeen, ID; the Oregon State University Malheur Experiment Station near Ontario, OR; and in a field near Paterson, WA. Variety tolerance trials were conducted in 2000, 2001, and 2002 at Aberdeen, ID, and in 2001 and 2002 near Paterson, WA. All trials were kept weed-free with periodic hand-weeding during the growing season. In the variety tolerance trials, Russet Burbank, Russet Norkotah, Ranger Russet, and Shepody were tested all years in ID and WA, and Alturas and Bannock Russet were included in the 2001 and 2002 ID trials. The ID trials were conducted on a Declo loam (coarse-loamy, mixed, mesic, Xerollic Calciorthid) with pH 7.9 (2000), 8.4 (2001), or 8.1 (2002) and 1.2 to 1.3% O.M. The OR Russet Burbank trial was conducted on an Owyhee silt-loam (coarse-silty, mixed, mesic Xerollic Camborthids) with pH 7.0 and 1.5% O.M. The WA trials were conducted on a Quincy sand (Mixed, mesic Xeric Torripsamments) with pH 7.0 and 0.5% O.M. Potato seed pieces were planted each spring at 25-cm intervals in rows spaced 91 cm apart at ID or OR sites or 86 cm apart at WA. Planting, herbicide applications, and harvest dates are listed in Table 1.

The current registered sulfentrazone rate range for potato is 105 to 280 g/ha (Anonymous 2004b, 2004c). The lowest rate (105 g/ha) is recommended for use on coarse-textured soils with pH $>$ 7.0 and $<$1 to 3% O.M., or medium-textured soils with pH $>$ 7.0 and $<$1% O.M. The highest registered rate (280 g/ha) is only recommended for use on fine-textured soils with pH $<$ 7.0 and $>$3% O.M. Although the proposed flumioxazin use-rate for potato is 53 g/ha (L. Welch, personal communication), early potato efficacy trials have been conducted with 105 g/ha as the expected use-rate. In the Russet Burbank tolerance trials, sulfentrazone was applied at 105, 140, or 210 g/ha at all locations, and an additional treatment of 280 g/ha was included at ID and WA but not OR because of space limitations. Flumioxazin was applied at 53, 105, or 140 g/ha in Russet Burbank tolerance trials at ID and WA. In the variety tolerance trials, sulfentrazone at 105, 210, or 280 g/ha was applied at WA, and at 105 or 210 g/ha, but not 280 g/ha because of space limitations at the ID site. Flumioxazin was applied at 105 or 210 g/ha at ID, targeting 1× and 2× the lowest efficacious rate at ID, and similarly at 53 and 105 g/ha at WA with an additional treatment of 140 g/ha also included at WA.

Nontreated controls of each variety were included for comparison. Rows were hilled just before potato emergence (standard grower practice), and herbicides were applied after hilling, but before potato emergence, with a CO_2-pressurized backpack or bicycle sprayer in 164 or 187 L/ha H_2O and incorporated immediately after application with 1 to 2 cm sprinkler irrigation.

In the Russet Burbank tolerance trials, treatments were replicated four times in plots 4 rows wide. In the variety tolerance trials in 2000 at ID and the 2001 and 2002 trials at WA, treatments were replicated four times in a split block design with herbicides as main plots and potato varieties as subplots. In the 2001 and 2002 ID trials, varieties were the main plots and herbicides were the subplots. Each subplot was 2 or 4 rows wide in WA or ID, respectively. Plot length in all trials was 9.1 m. The trial areas received standard irrigation to maintain minimum soil water content of 65% field capacity during each growing season. The experimental areas were fertilized according to university recommendations before planting, and additional fertilizer was applied through the irrigation system throughout each growing season based on petiole analysis. Potatoes were vine-killed with chemical desiccants 2- to 3-wk before harvest.

Overall potato visual injury, consisting mainly of plant stunting and some leaf discoloration-blackening and malformation-crinkling, was rated periodically throughout each growing season on a scale of 0% = no injury to 100% = complete death. No injury ratings were taken in OR at 8 and 12 WAT. Because stunting was the main injury observed during visual injury ratings, and because previous researchers have used height to assess tolerance of other crops to sulfentrazone (Dayan et al. 1997; Hulting et al. 1997), potato plant height measurements (base of plant to meristem) on a total of 20 plants from the 2 center rows were conducted in the ID variety trials, and plant height reduction was calculated as a percentage of nontreated control plant height for each variety. Tubers were mechanically harvested approximately 2 to 3 wk after vine kill. Total tuber yield and yield by grade according to U.S. Department of Agriculture standards (Anonymous 1991) were determined for U.S. No. 1 (tubers weighing ≥113 g with no defects).

Data Analyses. An analysis of variance (ANOVA) was performed using PROC GLM (PC-SAS[3]). Arcsine transformations were used on percent visual crop injury and plant height reduction data when needed to mitigate the skewness of the data and to meet the requirements of normality for analysis. When transformations changed the results, nontransformed data are presented along with the statistical analysis results performed on the transformed data. Weed-free control data were not included in the crop injury data analysis and were included for tuber yield comparisons.

With the exception of the ID variety tolerance-trials conducted in 2000, 2001, or 2002, orthogonal contrasts were performed to determine the significance of variety, herbicide, rate effects, and interactions. If variety interactions were significant, data were sorted by variety, and then the herbicide by rate interaction was determined by contrasts for each variety. If there were no interactions, and rate effect was significant, trend contrasts were performed to determine if response was linear or quadratic. If there were no interactions, and variety or herbicide effect was significant, means were separated by Fisher's Protected LSD procedure ($P = 0.05$). Because only 2 rates were included in the ID variety tolerance trials, percentage of injury and height reduction means were separated by Fisher's Protected LSD procedure ($P = 0.05$), depending on interaction and effect significance. Data were combined over years and locations when possible.

RESULTS AND DISCUSSION

Potato crop injury caused by PRE-applied sulfentrazone was observed in trials at all locations and years and consisted mainly of stunting with some leaf discoloration-blackening, and/or leaf malformation-crinkling. Crop injury caused by PRE-applied flumioxazin was similar, but blackening discoloration of leaves was not as evident. Black discoloration occurred in the interveinal areas of potato leaves and usually involved ≤10% leaf area.

Russet Burbank Tolerance Trials—2000. Although injury resulting from sulfentrazone was ≤5% at OR and WA at 2 WAT, injury at ID was 18 to 34%, increasing linearly as sulfentrazone rate increased (Table 2). At 4

[3] PC-SAS software, SAS Institute, SAS Campus Drive, Cary, NC 27511.

Table 2. Visual crop injury 2, 4, 8, and 12 WAT[a] as a result of sulfentrazone preemergence applications to Russet Burbank potato at Aberdeen, ID; Ontario, OR; and Paterson, WA, in 2000.

	Crop injury[b]					
	2			8		
Sulfentrazone rate	ID	OR/WA	4	ID	WA	12 WAT
g/ha	%					
105	18	4	10	0	3	1
140	20	4	11	0	7	2
210	24	5	17	2	15	4
280	34	5	25	3	22	5
Contrasts[c]						
Sulfentrazone rate	**	NS	****	NS	**	NS
Linear rate response	**	NS	****	NS	***	NS
Quadratic rate response	NS	NS	*	NS	NS	NS

[a] Abbreviations: WAT, weeks after treatment; NS, not significant $P > 0.05$.

[b] The location by sulfentrazone rate interaction was not significant for crop injury data 4 or 12 WAT and the location by sulfentrazone rate was not significant for OR and WA injury 2 WAT so those data are combined over locations. The OR location did not have visual injury ratings at 8 and 12 WAT.

[c] $*P = 0.05$; $**P = 0.01$; $***P = 0.001$; $****P \leq 0.0001$.

Table 3. Visual crop injury 2, 4, 8, and 12 WAT[a] as a result of flumioxazin preemergence applications to Russet Burbank potato at Aberdeen, ID, and Paterson, WA, in 2000.

	Crop injury[b]					
	2			8		
Flumioxazin rate	ID	WA	4	ID	WA	12 WAT
g/ha	%					
53	10	1	4	0	4	1
105	13	3	6	2	10	3
140	19	5	20	5	19	5
Contrasts[c]						
Flumioxazin rate	**	NS	**	****	**	NS
Linear rate response	**	NS	**	****	**	NS
Quadratic rate response	NS	NS	*	**	NS	NS

[a] Abbreviations: WAT, weeks after treatment; NS, not significant. $P > 0.05$.

[b] The location by flumioxazin rate interaction was not significant for crop injury 4 or 12 WAT so those data are combined over locations.

[c] $*P = 0.05$; $**P = 0.01$; $***P = 0.001$; $****P \leq 0.0001$.

WAT, injury averaged across all locations was 10 to 25% and increased in a nonlinear manner as sulfentrazone rate increased. Injury was <5% at 8 WAT at ID whereas injury at WA was 3 to 22% and increasing in a nonlinear manner as sulfentrazone rate increased. By 12 WAT, injury at ID and WA was ≤5% regardless of rate.

Significant injury may have occurred later in the growing season at OR and WA than at ID because soil pH was lower at OR and WA (7.0) than at ID (7.9). Sulfentrazone is less mobile in lower pH soils than in higher pH soils (Grey et al. 1997), so herbicide movement to the potato root uptake zone may have required additional time and irrigation events in OR and WA than in ID.

There were no significant interactions or treatment effects for U.S. No. 1 and total tuber yield data (data not shown). Sulfentrazone-treated Russet Burbank U.S. No. 1 and total tuber yields were 37 to 38 MT/ha and 52 to 54 MT/ha, respectively, compared with nontreated control yields of 37 and 52 MT/ha, respectively.

Similar to injury caused by sulfentrazone, potato crop response to flumioxazin occurred earlier in the growing season in ID than in WA, but by 12 WAT, injury was ≤5% (Table 3). There were no significant interactions or treatment effects for U.S. No. 1 and total tuber yield data, and similar to the sulfentrazone tolerance trial results, flumioxazin applied PRE did not reduce Russet Burbank tuber yields compared with the nontreated controls (data not shown). U.S. No. 1 and total tuber yields were 22 to 25 MT/ha and 41 to 44 MT/ha, respectively.

Variety Tolerance Trials. There were significant location by year by herbicide treatment and/or variety interactions for all variables, and the 2001 and 2002 ID experimental design was different than the 2000 ID and 2001 and 2002 WA design. Therefore, separate analyses were performed on data from each of the trial locations and years.

Idaho—2000. Injury ranged from 5 to 25%, depending on the variety, at 5 WAT, with injury caused by 105 g/ha similar to injury by 210 g/ha regardless of variety (Table 4). However, Russet Burbank and Russet Norkotah plant height reductions at 5 WAT from 210 g/ha were greater than reductions caused by 105 g/ha (Table 4). At 9 WAT, increasing the sulfentrazone rate from 105 to 210 g/ha increased injury for all varieties, and Russet Burbank injury from 210 g/ha was 22% compared with 7, 9, or 11% injury to Russet Norkotah, Ranger Russet, or Shepody, respectively (Table 4). Russet Norkotah was the only variety with greater height reduction caused by 210 than by 105 g/ha, however.

There were no significant variety by sulfentrazone interactions for tuber yields, and the sulfentrazone rate effect was not significant; therefore, initial injury and plant height reduction caused by sulfentrazone in the 2000 variety tolerance trial did not translate to tuber yield reductions (data not shown). As would be expected, varieties yielded differently, and U.S. No. 1 and total tuber yields were greater for Ranger Russet (34 and 42 MT/ha) or Shepody (35 and 43 MT/ha) than for Russet Norkotah (19 and 27 MT/ha) or Russet Burbank (23 and 33 MT/ha).

Table 4. Visual crop injury and potato height reduction 5 and 9 WAT[a] as a result of sulfentrazone applied PRE to four potato varieties in a weed-free trial at Aberdeen, ID, in 2000.[b]

	Crop injury[c]							
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Sulfentrazone rate	5	9	5	9	5	9	5	9 WAT
g/ha	%							
105	15 a	11 b	7 a	2 b	5 a	4 b	10 a	5 b
210	25 a	22 a	10 a	7 a	8 a	9 a	13 a	11 a
	Plant height reduction[d]							
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Sulfentrazone rate	5	9	5	9	5	9	5	9 WAT
g/ha	%							
105	11 b	18 a	19 a	5 a	16 b	5 b	16 a	6 a
210	20 a	23 a	14 a	9 a	30 a	20 a	17 a	8 a

[a] Abbreviations: WAT, weeks after treatment; PRE, preemergence; LSD, least significant difference.

[b] The variety by sulfentrazone rate interaction was significant for 5 and 9 WAT.

[c] Means within a column followed by the same letter were not significantly different according to a Fisher's Protected LSD test (P = 0.05) performed on arcsine-transformed data.

[d] Height of 20 plants total from the two center rows of each plot were measured. Plant height reduction is shown as percentage of reduction of treated plant height compared with nontreated control plant height. Means within a column within a variety followed by the same letter were not significantly different according to a Fisher's Protected LSD test (P = 0.05) performed on arcsine-transformed data.

Idaho and Washington—2001. At ID, injury caused by flumioxazin and sulfentrazone at 2 WAT was similar (9% and 8%), whereas injury caused by sulfentrazone was greater than injury caused by flumioxazin at 5 WAT (15 and 10%, respectively) and slightly greater at 9 WAT (9 and 7%, respectively) (data not shown in tables). Injury caused by 210 g/ha (10 to 15%) was greater than injury from 105 g/ha (6 to 10%) at all rating dates (Table 5). The effect of herbicide treatments on varieties is shown in Table 6. All varieties were injured similarly at 2 WAT, and averaged across herbicides and rates, injury ranged from 6 to 12%. At 5 WAT, Russet Norkotah (6%) was injured less than all other varieties (8 to 17%). By 9 WAT, injury to all varieties was ≤11%.

At 5 WAT, unlike visual injury, no herbicide and/or rate consistently caused height reduction at ID (Table 7). In general however, Russet Norkotah (1 to 8%) and Shepody (7 to 11%) suffered less numeric height reduction than the other varieties (9 to 32%). By 9 WAT, height reduction for all varieties except Russet Norkotah ranged from 4 to 15% and, in general, was numerically less than at 5 WAT. However, Russet Norkotah height reduction was 12 to 20% at 9 WAT compared with 1 to 8% at 5 WAT. Similar to visual injury, at 9 WAT the herbicide and/or rate causing the most height reduction within variety was not always the same as at 5 WAT.

At WA in 2001, averaged across variety and herbicide, injury at 2 WAT increased in a nonlinear manner from

Table 5. Visual crop injury as a result of flumioxazin and sulfentrazone preemergence applications to six potato varieties in a weed-free trial at Aberdeen, ID, in 2001.[a]

	Crop injury[b] 2001		
Rate	2	5	9 WAT[c]
g/ha	%		
105	7 b	10 b	6 b
210	10 a	15 a	10 a

[a] There were no significant interactions, and the rate effect was significant for data 2, 5, and 9 WAT. Data are shown averaged across herbicides and varieties.

[b] Means within a column followed by the same letter were not significantly different at P = 0.05 according to a Fisher's Protected LSD test performed on arcsine-transformed data.

[c] Abbreviations: WAT, weeks after treatment; LSD, least significant difference.

Table 6. Visual crop injury as a result of flumioxazin and sulfentrazone preemergence applications to six potato varieties in a weed-free trial at Aberdeen, ID, in 2001.[a]

	Crop injury[b]		
Variety	2	5	9 WAT[c]
Russet Burbank	9 a	16 a	11 a
Ranger Russet	10 a	17 a	10 ab
Russet Norkotah	6 a	6 c	6 ab
Shepody	8 a	8 ab	6 ab
Alturas	8 a	11 ab	4 b
Bannock Russet	12 a	17 a	11 a

[a] There were no significant interactions, and the variety effect was significant for data 2, 5, and 9 WAT. Data are shown averaged across herbicides and rates.

[b] Means within a column followed by the same letter were not significantly different at P = 0.05 according to a Fisher's Protected LSD test performed on arcsine-transformed data.

[c] Abbreviations: WAT, weeks after treatment; LSD, least significant difference.

Table 7. Potato plant height reduction 5 and 9 WAT[a] as a result of flumioxazin and sulfentrazone preemergence applications to six potato varieties in weed-free trials at Aberdeen, ID, in 2001 and 2002.[b]

Variety	Herbicide	Rate	Plant height reduction[c] 2001 5	2001 9	2002 5	2002 9WAT
		g/ha	%			
Russet Burbank	F	105	12 b	11 a	10 ab	15 a
	F	210	18 ab	6 b	11 ab	11 a
	S	105	14 b	9 ab	7 b	11 a
	S	210	21 a	6 b	13 a	15 a
Ranger Russet	F	105	12 bc	15 a	6 bc	8 a
	F	210	18 a	13 a	19 a	4 a
	S	105	15 ab	11 ab	11 b	13 a
	S	210	9 c	7 b	5 c	11 a
Russet Norkotah	F	105	1 b	13 b	18 b	5 c
	F	210	2 b	20 a	23 a	12 ab
	S	105	8 a	14 ab	15 b	7 bc
	S	210	8 a	12 b	20 a	14 a
Shepody	F	105	7 a	7 b	7 c	14 a
	F	210	9 a	5 b	16 a	14 a
	S	105	11 a	14 a	11 bc	14 a
	S	210	9 a	8 b	12 b	18 a
Alturas	F	105	13 a	5 b	11 b	14 a
	F	210	13 a	10 a	24 a	13 a
	S	105	12 a	4 b	10 b	15 a
	S	210	10 a	6 b	12 b	15 a
Bannock Russet	F	105	15 bc	7 a	14 a	12 c
	F	210	13 c	8 a	17 a	19 ab
	S	105	32 a	7 a	5 b	15 bc
	S	210	21 b	7 a	18 a	24 a

[a] Abbreviations: WAT, weeks after treatment; F, flumioxazin; S, sulfentrazone; LSD, least significant difference.

[b] The year by herbicide by rate by variety interaction was significant for height reduction 5 and 9 WAT. The herbicide by rate by variety interaction was significant for data 5 and 9 WAT in 2001 and 2002.

[c] Height of 20 plants total from the two center rows of each plot were measured. Plant height reduction is shown as percentage of reduction of treated plant height compared with nontreated control plant height. Means within a column within a variety followed by the same letter were not significantly different at P = 0.05 according to a Fisher's Protected LSD test performed on arcsine-transformed data.

4 to 12% as rate increased (data not shown). At 4 WAT, injury resulting from flumioxazin applied PRE to Russet Burbank or Ranger Russet was 2 to 16% or 2 to 20%, respectively, and increased linearly as the rate increased from 53 to 140 g/ha (Table 8). All rates injured Shepody (7 to 11%) and Russet Norkotah (6 to 13%) similarly at 4 WAT. By 9 WAT, Russet Burbank injury was numerically less (0 to 9%), but still increasing linearly as flumioxazin rate increased, whereas injury to Ranger Russet remained relatively high at 8 to 18%, increasing nonlinearly as flumioxazin rate increased. Shepody injury at 9 WAT was 1 to 13%, increasing linearly as flumioxazin rate increased, whereas all rates were still affecting Russet Norkotah similarly and injury ranged from 3 to 11%.

As sulfentrazone rate increased, injury to all four varieties at 4 WAT went from 3 to 16%, increasing in a linear manner as sulfentrazone rate increased from 105 to 280 g/ha (Table 8). At 9 WAT, injury from sulfentrazone to all four varieties also increased in a linear manner as rate increased. However, Russet Burbank and Russet Norkotah injury only ranged from 2 to 14%, whereas Ranger Russet and Shepody injury was as high as 27% and 25%, respectively.

Even though the varieties were injured differently by the different herbicides and rates tested at ID in 2001, there were no significant interactions for U.S. No. 1 and total tuber yield data (Table 9). Similar to 2000, the herbicide effect was not significant whereas the variety effect was significant (data not shown). Ranger Russet, Russet Norkotah, and Bannock Russet had greater U.S. No. 1 tuber yields (19 to 22 MT/ha) than the other three varieties (10 to 13 MT/ha), and Ranger Russet had greater total tuber yields than the other five varieties (37 and 28 to 32 MT/ha, respectively).

Unlike results in 2000, tuber yields were reduced as herbicide rate increased from 0 to 210 g/ha in 2001 (Table 9). Regardless of how much or little each variety was affected by herbicide and/or rate at ID during the 2001 growing season, and even though in general, injury to all varieties was less at 9 WAT compared with earlier

Table 8. Visual crop injury 4 and 9 WAT[a] as a result of flumioxazin and sulfentrazone preemergence applications to four potato varieties in weed-free trials at Paterson, WA, in 2001.[b]

	Crop injury							
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Flumioxazin rate	4	9	4	9	4	9	4	9 WAT
g/ha	%							
53	2	0	8	2	6	3	7	1
105	7	3	6	4	4	3	9	3
140	16	9	18	20	13	11	11	13
Contrasts[c]								
Rate effect	**	*	*	**	NS	NS	NS	**
Linear rate effect	**	**	*	**	NS	NS	NS	**
Quadratic rate effect	NS	NS	*	NS	NS	NS	NS	NS
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Sulfentrazone rate	4	9	4	9	4	9	4	9 WAT
g/ha	%							
105	4	2	7	5	3	3	7	5
210	9	7	9	12	4	6	11	13
280	14	10	16	27	10	14	13	25
Contrasts[c]								
Rate effect	**	**	***	***	**	***	*	**
Linear rate effect	**	**	***	****	**	***	**	**
Quadratic rate effect	NS	NS	NS	NS	NS	NS	NS	NS

[a] Abbreviations: WAT, weeks after treatment; NS, not significant, $P > 0.05$.

[b] The variety by herbicide by rate was not significant for data 4 WAT and was significant for 9 WAT. The variety by herbicide interaction was significant for data 4 and 9 WAT. The herbicide by rate interaction for data 4 WAT and the variety by rate interaction for 9 WAT were significant.

[c] *$P = 0.05$; **$P = 0.01$; ***$P = 0.001$; ****$P \leq 0.0001$.

in the growing season, that injury translated to yield reductions of all six varieties tested compared with the nontreated controls.

Variety, herbicide, and rate interactions were significant for tuber yields at WA in 2001, and the relatively high injury caused by flumioxazin or sulfentrazone to Ranger Russet or Shepody still observable at 9 WAT, translated to reduced tuber yields of those varieties compared with the nontreated controls (Table 10). Russet Burbank and Russet Norkotah, however, apparently recovered from initial injury because tuber yields of those varieties were not reduced compared with the nontreated controls. Ranger Russet U. S. No. 1 and total tuber yields decreased linearly as the rate of either herbicide increased, whereas Shepody total, but not U.S. No. 1, tuber yields decreased linearly as sulfentrazone rate increased from 0 to 280 g/ha. Increasing the flumioxazin rate from 0 to 140 g/ha did not affect Shepody tuber yields.

Table 9. U. S. No. 1 and total tuber yields as a result of flumioxazin and sulfentrazone preemergence applications to six potato varieties in a weed-free trial at Aberdeen, ID in 2001.[a]

	Tuber yield[b]	
Rate	U.S. No. 1	Total
g/ha	MT/ha	
0	19.8	33.3
105	17.1	29.9
210	17.2	29.9
Contrasts[c]		
Rate effect	****	****
Linear rate effect	****	****
Quadratic rate effect	**	**

[a] There were no significant interactions for yield data and the variety and herbicide effects were not significant. The rate effect was significant and data are shown averaged over varieties and herbicides.

[b] U.S. No 1 tubers have no defects and weigh ≥113 g.

[c] NS, not significant; $P > 0.05$; *$P = 0.05$; **$P = 0.01$; ***$P = 0.001$; ****$P \leq 0.0001$.

Idaho and Washington—2002. At ID, flumioxazin or sulfentrazone applied PRE at 105 g/ha resulted in ≤4% regardless of rating time (data not shown in tables). Injury caused by 210 g/ha compared with 105 g/ha was greater at all rating dates and was as high as 21% at 5 WAT but only 4% earlier and later in the season at 2 and 9 WAT. Plant height reduction ranged from 5 to 24% at 5 and 9 WAT, and as in 2000 and 2001, height of each variety was reduced differently by different herbicide treatments in 2002 (Table 7).

At WA in 2002, injury was <10% at 2 WAT, regardless of herbicide (data not shown). Injury caused by flumioxazin at 4 and 9 WAT also was <10%; however, sulfentrazone caused 2 to 24% and 1 to 20% injury at 4 and 9 WAT, respectively, with injury increasing linearly as rate increased (Table 11).

Unlike 2001 tuber yield results and even though injury

Table 10. Tuber yields as a result of flumioxazin and sulfentrazone preemergence applications to four potato varieties in weed-free trials at Paterson, WA, in 2001.[a]

	Tuber yields[b]							
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Flumioxazin rate	U.S. No. 1	Total	U.S. No. 1	Total	U.S. No. 1	Total	U.S. No. 1	Total
g/ha	Ml/ha							
0	59.4	71.1	72.5	79.5	43.0	53.6	55.6	62.8
53	61.1	74.7	63.2	72.1	47.9	58.1	47.9	54.9
105	59.2	73.1	65.1	73.2	49.3	59.0	47.7	59.0
140	58.4	69.4	58.6	66.8	51.0	61.0	52.3	57.8
Contrasts[c]								
Rate effect	NS	NS	*	*	NS	NS	NS	NS
Linear rate effect	NS	NS	**	*	NS	NS	NS	NS
Quadratic rate effect	NS	NS	NS	NS	NS	NS	NS	NS
	Russet Burbank		Ranger Russet		Russet Norkotah		Shepody	
Sulfentrazone rate	U.S. No. 1	Total	U.S. No. 1	Total	U.S. No. 1	Total	U.S. No. 1	Total
g/ha	MT/ha							
0	59.4	71.1	72.5	79.5	43.0	53.6	55.6	62.8
105	61.2	74.1	64.3	73.2	49.3	59.6	48.6	55.1
210	58.1	72.3	65.0	73.1	48.9	58.9	49.0	58.8
280	61.0	73.6	55.8	64.1	49.9	59.6	49.3	54.5
Contrasts[c]								
Rate effect	NS	NS	***	***	NS	NS	NS	*
Linear rate effect	NS	NS	***	****	NS	NS	NS	*
Quadratic rate effect	NS	NS	NS	NS	NS	NS	NS	NS

[a] The variety by herbicide by rate and the herbicide by rate interactions were not significant and the variety by herbicide and variety by rate interactions were significant for tuber yield.

[b] U.S. No 1 tubers have no defects and weigh $\geq$113 g.

[c] NS, not significant; $P > 0.05$; *$P = 0.05$; **$P = 0.01$; ***$P = 0.001$; ****$P \leq 0.0001$.

and height reductions were >20% earlier in the 2002 growing season, herbicide treatments did not reduce tuber yields compared with the nontreated controls at ID or WA in 2002 (data not shown). As in 2000, some varieties produced greater tuber yields than others (data not shown in tables). At ID in 2002, Ranger Russet, Russet Norkotah, Alturas, and Bannock Russet U.S. No. 1 tuber yields (36 to 41 MT/ha) were greater than Russet Burbank and Shepody U.S. No. 1 tuber yields (27 to 29 MT/ha) whereas Russet Burbank and Bannock Russet total tuber yields (46 to 48 MT/ha) were greater than total tuber yields of all other varieties (41 to 43 MT/ha).

Ranger Russet U.S. No. 1 tuber yields (66 MT/ha) were greater than U.S. No. 1 tuber yields of the other three varieties tested at WA in 2002 (40 to 59 MT/ha), and Russet Burbank and Ranger Russet total tuber yields (76 and 79 MT/ha, respectively) were greater than Russet Norkotah and Shepody total tuber yields (57 and 67 MT/ha, respectively).

Overall, PRE-applied flumioxazin or sulfentrazone caused potato crop injury at all locations in all years, however, yield reduction compared with nontreated controls only occurred in the 2001 trials at ID and WA. Crop response to the herbicides may have been different in 2001 compared with response in the other trial years because ID and WA experienced unusually high temperatures at the same time herbicide injury was observed and at the same time as tuber set, at or just before row closure in 2001. Li et al. (2000b) determined that increased temperatures coupled with increased sulfentrazone concentrations in a soybean seed imbibition solution resulted in greater soybean seedling height reduction. Heat-stressed potato plants exposed to sulfentrazone, especially during a crucial developmental period, such as tuber set, may have been more affected by the herbicide in 2001 compared with other years when heat stress did not occur.

Taylor-Lovell et al. (2001) proposed that a reduction in flumioxazin or sulfentrazone metabolism occurred when soybeans were subjected to an environmental stress in their study. More severe soybean injury resulted from herbicide treatments in a year with cooler emergence temperatures compared with injury occurring during a year with warmer emergence temperatures. Similarly, potato plants in our trials may not have been able to tolerate heat stress occurring at the same time as other proposed herbicide stresses, such as protoporphyrin IX-induced peroxidative stress (Dayan et al. 1997).

Table 11. Visual crop injury and tuber yield as a result of flumioxazin and sulfentrazone preemergence applications to four potato varieties in a weed-free trial at Paterson, WA, in 2002.[a]

Flumioxazin rate	Crop injury		Sulfentrazone rate	Crop injury	
	4	9 WAT[b]		4	9 WAT
g/ha	%		g/ha	%	
53	1	0	105	2	1
105	4	2	210	11	9
140	9	2	280	24	20
Contrasts[c]					
Rate effect	****	NS		****	****
Linear rate effect	****	NS		****	****
Quadratic rate effect	NS	NS		NS	NS

[a] The variety by herbicide by rate, variety by rate, and variety by herbicide interactions were not significant for data 4 and 9 WAT. The herbicide by rate interaction was significant for data 4 and 9 WAT.

[b] Abbreviations: WAT, weeks after treatment.

[c] NS, not significant; $P > 0.05$; *$P = 0.05$; **$P = 0.01$; ***$P = 0.001$; ****$P \leq 0.0001$.

Soil pH was higher at the 2001 ID field site (8.4) compared with the 2000 (7.9) and 2002 (8.1) ID sites. Reports of higher soil pH possibly increasing sulfentrazone phytotoxicity have been made (Wehtje et al. 1997). Because sulfentrazone is more mobile in soils with higher pH than in soils with lower pH, movement of greater herbicide concentration to the potato root zone with irrigation water could have occurred at ID in 2001 compared with 2000 or 2002.

In addition, as a result of high temperatures occurring unusually early in the 2001 growing season, both 2001 trial sites generally received more frequent irrigation events between the time of potato emergence and row closure compared with other trial years. Price et al. (2004b, 2004c) have stated that rainfall before crop emergence would move flumioxazin from the soil surface into the soil profile, preventing crop injury from rain splash after crop emergence. However, irrigation or excessive moisture or rainfall, especially when experienced shortly after crop emergence, has enhanced flumioxazin and sulfentrazone injury and has caused yield reductions in soybeans and other crops as well (Burke et al. 2002; Grichar et al. 2003; Krausz and Young 2003; Swantek et al. 1998; Taylor-Lovell et al. 2001; Teuton et al. 2004).

Li et al. (2000b) have speculated that rainfall occurrence shortly after sulfentrazone application increases the amount available for seedling absorption in soil solution. Increased total irrigation amount between the time of potato emergence and row closure at ID in 2001 most likely moved the sulfentrazone more quickly and in higher concentrations to the potato root uptake zone than in 2000 or 2002. Increased sulfentrazone concentration in the root uptake zone may have caused more herbicide-induced stress than in other years and, compounded by heat stress, may be the reason all six varieties suffered tuber yield loss compared with the nontreated controls in 2001 but not in the other years.

Movement of flumioxazin to the potato root zone was probably similar at all ID sites during all trial years because the percentage of O.M. was similar at those sites (1.3 to 1.4%). However, wet and/or cold conditions encountered after PRE applications of flumioxazin have facilitated injury to peanut (*Arachis hypogaea* L.) (Burke et al. 2002; Teuton et al. 2004) and soybean (Niekamp et al. 1999; Sakaki et al. 1991), therefore, increased irrigation in our 2001 trial could have similarly caused increased flumioxazin uptake, and heat stress rather than cold temperatures may have slowed flumioxazin metabolism.

Of the four varieties tested at WA in 2001, sulfentrazone applied PRE at 105 to 280 g/ha only caused reductions in tuber yields of Ranger Russet and Shepody, whereas flumioxazin at 53 to 140 g/ha only caused tuber yield reductions of Ranger Russet compared with the nontreated controls. Russet Burbank and Russet Norkotah tuber yields were unaffected by flumioxazin or sulfentrazone at WA. These results are in contrast to ID results in 2001 when all four varieties, plus two additional varieties, suffered tuber yield reductions resulting from flumioxazin or sulfentrazone applied PRE at 105 or 210 g/ha. Even though the highest flumioxazin rate at WA was only 140 g/ha, greater concentrations of flumioxazin still may have been available for uptake and subsequent injury at WA compared with ID because the WA soil had a lower percentage of O.M. than the ID soil—0.5% compared with 1.3 to 1.4%.

As mentioned, heat stress occurred approximately the same time as tuber initiation in 2001. Tuber initiation, sometimes called tuber set, usually occurs over a period of approximately 2 wks when stolon tips begin to swell, resulting in initiation of a new tuber (Dwelle 2003; Rowe and Secor 1993). The two major factors influencing potato tuber yield are (1) the photosynthetic activity and duration of the leaf canopy, and (2) length of time of the tuber growth or bulking phase (Dwelle 2003). Stress occurring during the period when tuber set would normally occur can cause a delay in tuber set, consequently decreasing the time for tuber bulking before season-end and, ultimately, reducing tuber quality and yield. Any reduction in photosynthetic activity during the beginning of the tuber bulking phase, which may have occurred if these protox-inhibiting herbicides affected chlorophyll production in the potato plants detrimentally, also would effect tuber quality and yield.

Differential response of Ranger Russet and Shepody compared with the other potato varieties tested in the 2001 WA trial may be attributed to reasons similar to aforementioned differential soybean cultivar response to these herbicides. However, Ranger Russet may have been more affected than the other varieties in WA because tubers initiating on Ranger Russet plants generally initiate higher/closer to the soil surface on the below-ground root system than where tubers initiate on the other varieties (S. Love, personal communication). Once tubers initiate, they become the dominant meristems and sinks in the potato plant (Moorby 1978).

Sulfentrazone movement downward in the soil profile may have been relatively slow in WA because the soil pH of 7.0 was near the sulfentrazone pKa of 6.56, and not much of the herbicide was likely in the anionic, more mobile form. Therefore, early in the 2001 season, during that heat stress and tuber set period, Ranger Russet tubers initiating closer to the soil surface could have been exposed to higher sulfentrazone concentrations than tubers of the other varieties initiating lower in the soil profile because much of the uncharged herbicide may not have moved far from the initial incorporation depth near the soil surface.

Similarly, although flumioxazin mobility was most likely less affected than sulfentrazone mobility because the WA soil only had 0.5% O.M., a greater flumioxazin concentration was probably present near the tubers initiating on the Ranger Russet plants higher in the soil profile than the herbicide concentration near the tubers initiating lower in the profile on the other varieties. Also, because tubers growing on the other varieties tested in WA may not have been exposed to flumioxazin until slightly later in the season, perhaps, as with cotton (Price et al. 2004a), increased metabolism by the later-season, more mature potato plants resulted in less herbicide-induced stress than what occurred in younger Ranger Russet plants earlier in the season.

Because of the lower elevation, WA usually experiences a longer growing season than ID. In 2001, however, the time interval between planting and harvest was shorter in WA than in other trial years and similar to the length of the growing season in ID in all trial years (Table 1). The relatively shorter growing season coupled with heat and herbicide stress-induced injury to Ranger Russet and Shepody resulted in yield losses when those injured potato plants may have recovered by the end of a longer growing season.

The other varieties tested in WA in 2001 were able to recover from initial injury, even during a short growing season, possibly because of differential varietal metabolism, less root absorption, and/or initiating tuber exposure to lower herbicide concentrations at the same time as heat stress. Consequently, herbicide injury was not compounded by other factors, and yields were not ultimately reduced in those varieties.

In all trial years, injury generally occurred later in the growing season relative to herbicide application dates in WA than in ID. Even though the soil at the WA site was more coarse-textured and had less O.M., and therefore, less potential herbicide adsorption capacity than soil at the ID sites, soil pH in WA (7.0) was lower and closer to the sulfentrazone pKa than soil pH in ID (7.9 to 8.4). Less sulfentrazone would be in the anionic form in the WA soil compared with the ID soils, less sulfentrazone would be repelled by positively charged sites on soil colloids/organic matter, and consequently, sulfentrazone movement to the potato root uptake zone in WA may have been slower and in lower concentrations than what occurred in ID. Consequently, injury occurred later at WA than at ID because herbicide movement to the potato root uptake area required more time and additional irrigation events at WA.

These observations are in agreement with other researchers reporting that the variable pH-charge dependency of sulfentrazone affects its overall ionization, chemical activity, and fate in soil (Grey et al. 2000b) and that soil pH is a more important factor than soil type when considering influence on sulfentrazone mobility and availability (Grey et al. 1997). Kerr et al. (2004), however, observed less sunflower (*Helianthus annuus* L.) response to sulfentrazone from increasing pH than was anticipated, and contrary to conclusions by Grey et al. (1997), they concluded that soil type, as indicated by cation-exchange capacity, had a greater effect than soil pH on sunflower injury caused by sulfentrazone.

Grey et al. (1997) conducted sulfentrazone mobility and availability research using radiotracer technology and soil thin-layer chromatography in a laboratory setting, whereas observations made by Kerr et al. (2004) were based on results from a sunflower plant bioassay conducted in the greenhouse. Although the latter study involved a crop, seed placement and root zone depth were most likely uniform in their potted-soils. Many factors dependent on herbicide mobility and availability existed in our trials, such as varietal differences in potato tuber-set depth and plant-maturity at time of herbicide arrival at root uptake areas. Even though soil pH was lower at WA than at ID, the pH at both locations was greater than the sulfentrazone pKa resulting in the presence of at least some sulfentrazone in the anionic form. Intensive irrigation is necessary for successful potato production in the semi-arid conditions of the PNW, and with the impact of irrigation on sulfentrazone movement in our trials, soil pH played a more important role in potato crop response to sulfentrazone than soil type. In addition, although Ferrell et al. (2003) determined that sulfentrazone absorption by plant roots increases as soil or solution pH decreases, sulfentrazone mobility and availability as affected by the high soil pH seemed to be a key factor in the occurrence and possible severity of the potato crop response in our trials.

In our experiences, when potato growers observe crop injury during the growing season, they usually become quite concerned about potential tuber quality and yield reductions. Flumioxazin or sulfentrazone potato crop injury symptoms, such as stunting, chlorosis, necrosis, and/or leaf-crinkling, could be expected because these symptoms have been observed on soybean and other crops treated with flumioxazin or sulfentrazone (Askew et al. 2002; Burke et al. 2002; Dayan et al. 1996; Kerr et al. 2004; Main et al. 2003, 2004; Price et al. 2004a, 2004c; Swantek et al. 1998; Taylor-Lovell et al. 2001; Teuton et al. 2004; Wehtje et al. 1995; Wilcut et al. 2001; Wilson et al. 2002). Reductions in hypocotyl and root elongation have occurred in sulfentrazone-treated soybeans (Li et al. 1999), and soybean height reduction has been used as an indicator of soybean susceptibility to sulfentrazone (Dayan et al. 1997; Hulting et al. 1997).

The injury symptom of leaf-discoloration with interveinal black areas on leaves observed in our potato tolerance trials has not been previously reported, however, and was unexpected. This symptom has subsequently been observed at greater levels in 2004 ID PRE and postemergence sulfentrazone/potato research trials (unpublished data) and in commercial potato fields where sulfentrazone was applied PRE. Early-season temperatures were cooler than normal in most ID potato production areas, and unusual, significant amounts of rainfall occurred up to 1 mo after sulfentrazone application in many ID potato fields in 2004. Occurrence of this symptom, seemingly unique to potatoes, warrants further investigation of sulfentrazone effect on potatoes, especially with regard to protox inhibition in potato, potato tolerance mechanisms, and possible reactions of potato to accumulated chlorophyll intermediates and/or to herbicide-induced peroxidative stress.

Sulfentrazone or flumioxazin applied PRE resulted in early-season potato crop injury during all three trial years, but only caused subsequent tuber yield reductions in 2001. The occurrence of potato crop injury without tuber yield reductions in our 2000 and 2002 trials is similar to results of other potato tolerance trials when injury caused by flumioxazin or sulfentrazone did not translate to tuber yield reductions (Bailey et al 2002; Kazarian et al 2001; Wilson et al 2002), and when yield reductions in other crops did not occur even though these herbicides caused crop injury earlier in the growing season (Askew et al. 2002; Burke et al. 2002; Dayan et al. 1997; Ferrell et al. 2003; Grey et al. 1997; Grey et al. 2000a; Krausz and Young 2003; Main et al. 2003; Miller 2003; Nolte and Young 2002; Price et al. 2004b, 2004c; Scott et al. 2001; Viator et al. 2002; Wilcut et al. 2001).

Crop injury did translate to tuber yield reduction 1 of 3 yrs in our trials. Although Grichar et al. (2003) reported that the only potato tuber yield reductions caused by sulfentrazone applied PRE occurred in a location in which irrigation was applied 48 h after application, and the soil type was more coarse than at other trial locations, they also stated that the most severely stunted potato plants in their sulfentrazone trials also produced less potatoes. Taylor-Lovell et al (2001) reported that soybean yield reductions appeared to be related to, but not as severe as, visible injury caused by flumioxazin or sulfentrazone, and Main et al. (2004) stated that carefully made visual evaluations in sulfentrazone trials closely correlated with cotton yields.

These contradictory observations of injury and reduced crop yield correlations and our varied results lead to a feasible conclusion that if potato plants exposed to sulfentrazone or flumioxazin exhibit injury symptoms, yield reductions still may not occur. However, injury may translate to tuber yield reductions if that injury causes delayed development during a short-growing season or is compounded by environmental stress; lack of tolerance due to inherent, varietal traits; exposure and uptake when young potato plants may not be able to metabolize the herbicide as well as older, more mature plants; or at inopportune, critical developmental periods such as tuber initiation.

Soil pH seemingly affected herbicide mobility and availability resulting in more crop injury in our trials than soil type and/or organic matter. Irrigation also appeared to have a significant effect. Conditions conducive to herbicide movement to the potato root uptake and/or tuber initiation zone, or too much irrigation or rainfall occurring soon after application may result in herbicide injury and subsequent potato tuber yield loss.

When other stress-inducing factors, such as unusually high or low temperatures, occur at the same time as herbicide-induced stresses, potato plants may not be able to recover from minimal herbicide damage in short growing-season regions, regardless of variety, and therefore, yields may be affected detrimentally. Some varieties may not be able to recover from flumioxazin or sulfentrazone injury even in longer growing-season regions when herbicide stress coupled with environmental stress occurs during crucial potato development periods. Appropriate herbicide rate ranges based on differences in injury between locations with differing soil types, percentages of O.M., and especially soil pH, should be more completely developed for use of flumioxazin and sulfentrazone in irrigated potato production areas.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the technical assistance of Felix E. Fletcher, Joey K. Ishida, and Marcus Seymour. This research was supported in part by the Idaho Potato Commission, FMC Corporation, and Valent USA Corporation.

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